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Acute tubular necrosis induced by coronary thrombolytic therapy

Sir,

It is well recognized that streptokinase infusion may be accompanied by systemic hypotension, and indeed, occasionally cardiovascular collapse.¹ Cases of renal failure associated with the use of thrombolytic drugs can thus be anticipated. A 74 year old lady presented with an acute inferior myocardial infarction. She had a blood pressure of 110/70 mmHg, 1st degree heart block with a cardiac rate of 96/min and there was no evidence of left ventricular failure. Her renal biochemistry was within normal limits and her only medication was tranlycypromine. Streptokinase infusion (1 mega-unit over 1 hour) was commenced on the coronary care unit but was accompanied by severe hypotension (40 mmHg systolic) after 10 minutes, followed by a grand mal convulsion. Thrombolytic therapy was discontinued immediately and dobutamine infused, initially at a dose of 20 μ g/kg/min. Despite inotropic support the patient remained significantly hypotensive for 2 hours although the cardiac rhythm remained unchanged.

Acute oliguric renal failure developed over the following 48 hours and this necessitated a period of peritoneal dialysis treatment. Renal recovery was delayed and she was subsequently transferred to our unit for further management. Investigations, including a radio-labelled renal 1st circulation study, were compatible with a diagnosis of acute tubular necrosis, and there was also evidence of underlying narrowing of the left renal artery. The patient is now well but she still has moderate impairment of renal function.

Allergic side effects of streptokinase are well recognized and may include hypotension and a serum-sickness type reaction which has been observed in association with transient impairment of renal function.² Although significant hypotension (systolic blood pressure <80 mmHg) is observed in approximately 10% of patients with acute myocardial infarction who receive

streptokinase at standard infusion rate (1.5 million IU/h),³ the effect is usually short-lived (<10 minutes) and inotropic support rarely required.⁴ However, hypotension which is refractory to catecholamines may complicate the administration of streptokinase to patients with severe left ventricular dysfunction, and it has been recommended that such haemodynamically unstable subjects should receive the infusion at a much slower rate (200 IU/kg/min) – this perhaps should also be the case when treating some elderly patients. As similar hypotensive effects may complicate therapy with other thrombolytic agents, it is unlikely that a specific action of streptokinase is aetiologically important.⁴

We have shown that significant renovascular narrowing is a common finding in middle-aged and elderly patients who have evidence of generalized atheroma. Such renal artery pathology may be associated with hypertension or may remain clinically occult, particularly in elderly patients.⁵ These should be identified as the patients most likely to have kidneys that will be vulnerable to ischaemic damage if systemic hypotension supervenes. Efforts to minimize renal hypoperfusion in such patients are necessary if acute renal failure is to be avoided, and a slower rate of streptokinase infusion may need to be considered in the at-risk (especially elderly) population requiring coronary thrombolysis.

P.A. Kalra

A.M. Coady

A. Iqbal

A. Evans

S. Waldeck

Renal Unit, Hope Hospital

Eccles Old Road

Salford, UK.

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